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Murray, Aja Louise ; Booth, T ; Obsuth, I ; Zirk-Sadowski, J ; Eisner, Manuel ; Ribeaud, Denis

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Testing the exacerbation and attenuation hypotheses of the role of anxiety in the relation between ADHD and reactive/proactive aggression

Aja Louise Murray[1,2], Tom Booth[2], Ingrid Osbuth[3], Jan Zirk-Sadowski[4], Manuel Eisner[1] & Denis Ribeaud[5]

[1]Institute of Criminology, University of Cambridge, UK

[2]Department of Psychology, University of Edinburgh, UK

[3]School Health in Social Science, University of Edinburgh, UK

[4]Faculty of Medicine, University of New South Wales, Australia

[5]Jacobs Center for Productive Youth Development, University of Zurich, Switzerland

*Corresponding author at: Institute of Criminology, University of Cambridge, CB3 9DA; email: am2367@cam.ac.uk

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Abstract

Both anxiety and aggression commonly co-occur with ADHD symptoms. Two competing hypotheses describing the role of anxiety in aggression associated with ADHD symptoms have previously been advanced. The exacerbation hypothesis proposes that the presence of anxiety increases the risk of aggression in the context of ADHD symptoms. The attenuation hypothesis proposes that the presence of anxiety protects against aggression in the context of ADHD symptoms. We tested these hypotheses using moderated cross-lagged panel models in the Zurich project on social development from childhood to adulthood (z-proso) sample using both self-report (3 waves) and informant-report (8 waves) data spanning ages 7 to 17. We found evidence that anxiety protects against both reactive and proactive aggression; however, the effect was direct: there was no evidence for anxiety moderating the strength of ADHD symptom-aggression links. Results suggest that anxiety likely plays an important role in inhibiting aggression but does not interact with ADHD symptoms in the manner predicted by either the exacerbation or attenuation hypothesis.

Keywords: cross-lagged panel model, development, inattention, hyperactivity/impulsivity

1. Introduction

Attention-deficit hyperactivity disorder (ADHD) is characterised by significantly impairing levels of hyperactivity, impulsiveness and inattention (APA, 2013). Prevalence estimates vary widely across studies from less than 1% up to around 20% but centre on approximately 4-7% of the population meeting clinical diagnostic criteria for ADHD (Polanczyk et al., 2007; Polanczyk et al., 2014; Thomas et al., 2015). However, many others are affected at the sub-clinical level owing to the dimensional nature of ADHD symptoms (e.g. Groen-Blokhuis et al., 2014; Heidbreder et al. 2015; Hong et al., 2014; Lubke et al., 2009). ADHD symptoms predict a range of adverse outcomes, including economic disadvantage, criminality, poor academic performance, impaired social functioning and substance use (e.g. Dalsgaard et al., 2002; Frazier et al., 2007; Hong et al., 2014; Mannuzza et al., 2008).

At least some of the adverse outcomes linked to ADHD symptoms have their roots in associated behavioural problems, including aggression (e.g. Evans et al., 2016; Hinshaw, 1992; Waschbush and Willoughby, 1998). It is useful to delineate two broad types of aggression. First, reactive aggression refers to 'hot' emotion-driven responses to perceived threats or provocations. Second, proactive aggression refers to emotionally 'cold' instrumental behaviours. Much evidence supports the utility of this distinction. Although they are highly correlated, reactive and proactive aggression tend to emerge as separate dimensions in factor analyses (e.g. Murray, Eisner, et al., 2016; Raine et al. 2006), show distinct nomological networks (Card and Little, 2006; Cima and Raine, 2009), differ in their developmental trajectories (e.g. Cui et al., 2015; Dodge et al., 1997; Murray, Obsuth, et al., 2016) and show differential treatment responses (e.g. Barker et al., 2010).

The distinction between reactive and proactive aggression is also important in the context of ADHD symptoms. It is reactive aggression that appears to be most characteristic of the aggression observed in ADHD (e.g. Dodge and Coie, 1987). ADHD symptoms and reactive aggression are consistently correlated and this is assumed to reflect a direct influence of ADHD symptoms on reactive aggression (Becker et al., 2012; Bennett et al., 2004; Dodge et al. 1997; Bubier and Drabick, 2009; Retz and Rösler, 2009; Vitaro et al., 2002). In contrast, most theoretical perspectives posit only an indirect influence of ADHD symptoms on proactive aggression – via peer deviancy training – a process by which youth with ADHD are more likely to associate with and be influenced by antisocial peers (Bennet et al., 2004). Empirically they are correlated only inconsistently; both in terms of the presence of an association and its direction (e.g. Bennet et al., 2004; Card and Little, 2006; Retz and Rösler, 2010).

The proactive versus reactive aggression distinction may also be important for understanding the role of anxiety in the aggressive behaviour associated with ADHD symptoms. Anxiety symptoms are elevated and common in ADHD with around 25% of affected children experiencing significant anxiety (Angold et al., 1999; Kessler et al. 2012; Dallos, et al., 2014; Schatz and Rostain, 2006). Like ADHD symptoms themselves, an anxiety-ADHD correlation extends into sub-clinical symptoms in non-clinical populations (Baldwin and Dadds, 2008). According to two competing hypotheses: the exacerbation and attenuation hypotheses; anxiety symptoms have significant implications for ADHD-aggression links (Becker et al., 2012; Lilienfeld, 2003). The attenuation hypothesis holds that high levels of anxiety reduce aggressive behaviour associated with ADHD symptoms. In contrast, the exacerbation hypothesis holds that high levels of anxiety increase aggressive behaviour associated with ADHD symptoms. Evidence for these contradictory hypotheses is limited by the paucity of direct tests. Among the small number of studies that have reported

relevant data, results are mixed with respect to which, if either, is supported (e.g. Becker et al., 2012; Bloemsma et al., 2013; Falk et al., 2015; Sørensen et al., 2011). For example, Falk et al. (2015) found evidence consistent with attenuation. In a sample of 380 youth aged 5 to 17, they found that those with both ADHD and anxiety showed significantly lower levels of aggression than those with ADHD only. Humphreys, Aguirre and Lee (2012), however, found evidence consistent with exacerbation. In a sample of 203 6-9 year olds, they found that children with ADHD and comorbid anxiety had higher levels of oppositional defiant disorder and conduct disorder symptoms than those with either ADHD or anxiety alone. Becker et al. (2012) found evidence for neither exacerbation nor attenuation in two clinical youth samples (n=99 and n=265 aged between 6 and 12).

Greater clarity may be achieved and the apparent contradiction between the exacerbation and attenuation hypotheses potentially explained by consideration of the reactive versus proactive aggression distinction (e.g. Becker et al., 2012). Specifically, we here argue that the exacerbation hypothesis may describe the role of anxiety in the ADHD symptoms-reactive aggression relation while the attenuation hypothesis may describe its role in the ADHD symptoms-proactive aggression relation. The idea that anxiety exacerbates reactive aggression follows from the fact that this form of aggression is often driven by a sense of threat, fear or panic (Bubier and Drabick, 2009). Individuals high in anxiety may, therefore, experience heightened and more frequent impulses to aggress because they experience the world as generally more threatening. Those who are also high in ADHD symptomology are likely to face greater difficulties in managing these impulses (e.g. also see Barkley, 1997; DeWall et al., 2007). Thus, we would predict that the relation between anxiety and reactive aggression is stronger at higher levels of ADHD symptomology and, equivalently, that the relation between ADHD and reactive aggression should be stronger at higher levels of anxiety. On the other hand, as proactive aggression is premeditated,

individuals high in anxiety would be less likely to engage in proactive aggression where, in the absence of any emotional impulse to aggress, anxiety acts as a behavioural inhibitor. In this view, the relation between ADHD symptoms and proactive aggression should be attenuated at higher levels of anxiety because whatever risk for proactive aggression is conferred by ADHD symptoms should be tempered by the behavioural inhibition associated with anxiety.

There is currently little empirical evidence that can speak to these predictions directly. In fact, to our knowledge, only one study of the exacerbation and attenuation hypotheses has made a distinction between reactive and proactive aggression (Becker et al., 2012). However, this study used two clinical samples and although such samples have obvious clinical validity, they may be less well placed than community samples to detect and accurately quantify ADHD by anxiety interactions. In clinically ascertained samples there will be restricted variability in ADHD and anxiety symptoms which can lead to reduced power to detect already difficult-to-detect interactions (Murray et al., 2014; Shieh, 2009). Furthermore, the study was cross-sectional and can thus provide limited insight into developmental and causal relations between anxiety, ADHD and reactive and proactive aggression. It was, therefore, the aim of the current study to provide a test of the exacerbation and attenuation hypotheses in a large normative longitudinal sample using dimensional measures of ADHD, anxiety and reactive and proactive aggression.

2. Method

2.1. Participants

Participants were from the Zurich Project on the social development from childhood to adulthood (z-proso). Z-proso is a longitudinal cohort study focussing on the development of pro- and anti-social behaviour throughout childhood and adolescence. The study spans ages 7 to 17 in 9 measurement waves, with 7 waves at annual intervals followed by 2 waves at biennial intervals. The study began when the children entered school in 2004. They were invited to participate via their parents if they attended one of 56 Zurich-based schools selected using a stratified random sampling procedure that took into account geographical location and size. From the initial target sample size of 1675, 1359 (81%) of young people provided valid data on the variables of interest in the current study at intake (approximately 50% male). In being based in Zurich, the study sample is diverse in both ethnic and socioeconomic terms. At baseline, approximately 70 different nationalities were represented among the primary caregivers, with the biggest proportions of parents from Switzerland (38.4%), Italy (8.8%), Serbia-Montenegro (8.7%), Germany (6%) and Portugal (5%). At baseline (according to reports from parents on whom data was available) the highest educational levels of male primary caregivers were: 21% mandatory school or less, 35.2% apprenticeship, 7.8% A-levels (the highest level of school qualifications), 15.5% higher vocational education (a continuation of an apprenticeship), and 16% University. Based on a comparison of those who participated versus those who declined to participants, participants were mostly representative of the target sample with the exception that children of parents who did not speak German as a first language were slightly under-represented. From baseline, participant retention remained high, with around 1306 youths contributing data to the most recent wave (83% of the total recruited sample; 78% of the original target sample).

The study included separate child and parent intervention components in the early waves. There, however, was little evidence that they had substantive short or long-term effects (e.g. Averdijk et al., 2016; Malti et al., 2011). As such, it is not normally judged

necessary to statistically control for intervention participation or restrict analyses to those not exposed to the interventions. Further details of participant recruitment, characteristics and assessment procedures can be found in previous publications (e.g. Eisner and Ribeaud, 2007; Eisner et al., 2018) and on the z-proso website: http://www.jacobscenter.uzh.ch/en/research/zproso/aboutus.html.

2.2. Measures

We tested our hypotheses using both teacher-reported and self-reported data, obtained using the teacher and child versions of the Social Behavior Questionnaire (SBQ) (Tremblay et al., 1991). The SBQ is an omnibus psychopathology inventory measuring primarily internalising and externalising symptoms as well as pro-social behaviours. As administered to the participants of z-proso, the content of the SBQ was adjusted over the course of the 9 waves of data collection in order to maintain the developmental appropriateness of the questionnaire. In this study, we focused on the indicators of each construct that were consistent across all waves in order to facilitate as direct a comparison of behaviour over time as possible. As the teacher and child items did not correspond exactly, we analysed teacher and child data separately. This approach also avoids the blurring of potentially substantively important distinct features of self- versus teacher reports of symptoms (e.g. De Los Reyes, 2011). The cross-informant correlations at the two waves for which self- and teacher- reports overlapped (i.e., age 13 and 15) were low to moderate based on mean scores. The youth-teacher correlations for ADHD were 0.27 ($p<0.05$) at age 13 and $r=0.19$ ($r<0.05$) at age 15; for anxiety they were $r=0.15$ ($p<0.05$) and $r=0.13$ ($p<0.05$); for reactive aggression they were $r=0.25$ ($p<0.05$) and $r=0.23$ ($p<0.05$); and for proactive aggression they were $r=0.21$ ($p<0.05$) and $r=0.21$ ($p<0.05$). The reliability and validity of the SBQ as used in z-proso has been evaluated in several previous studies (e.g. Murray et al., 2017; Murray, Obsuth et al., 2018) which have supported the reliability, criterion validity, factorial validity and

developmental invariance of the SBQ. Given the challenge of maintaining developmental appropriateness but also comparability of items over such a wide age range as is represented in child development studies such as z-proso, much consideration has been given to optimising this trade-off (e.g. Murray, Obsuth et al., 2018). In brief, though the items were originally developed for use in younger age groups (e.g. Tremblay et al., 1991); previous studies in the current sample have provided support for the reliability and validity in late childhood and adolescence (e.g. Murray et al., 2017; Murray, Obsuth et al., 2018). Further, Murray, Obsuth et al. (2018) provided evidence that the items showed metric invariance across ages 11 (or 13 dependent on pattern of administration) to 17 suggesting that the items measure essentially the same constructs across the adolescent years.

2.2.1. Observer (teacher) measures

Informant (teacher) measures of anxiety, ADHD symptoms, reactive aggression and proactive aggression were available for 8 waves of data covering the entire age range of compulsory schooling in Zurich. Teachers provided ratings when the youths were aged approximately 7, 8, 9, 10, 11, 12, 13 and 15. Children usually had the same teachers between grades 1 to 3 (ages 7, 8 and 9) and between grades 4 to 6 (ages 10, 11, 12). At this point they then entered secondary school (ages 13 and 15). Anxiety was measured using three items referring to being nervous/tense, fearful/anxious and worried. ADHD symptoms were measured using four items measuring hyperactive/impulsive symptoms and four items measuring attention-deficit symptoms. The hyperactivity/impulsivity items referred to impulsivity, being impatient with turn-taking, being restless/hyperactive and fidgeting. The attention-deficit items refer to being unable to settle, distractibility, being unable to sustain attention and being inattentive. Reactive aggression was measured using three items referring to aggressive responding to teasing, being contradicted and having something taken from them. Proactive aggression was measured using 4 items referring to making threats,

encouraging bullying, trying to dominate others and scaring others. The scale was administered in a paper and pencil format in German, the official language of the canton of Zurich. Responses were measured on a five-point scale from *never* to *very often*.

2.2.2. Self-report (youth) measures

Self-report measures of anxiety, ADHD symptoms, reactive aggression and proactive aggression were available at 3 time points when the youths were aged approximately 13, 15 and 17. Self-report ratings were obtained using a self-report version of the SBQ. Items are worded similarly to those administered to teacher informants. Again, we focused on the items that were common to all three time points to maximise comparability. Anxiety was measured using three items referring to crying, worrying and feeling fear. ADHD symptoms were measured using four items referring to restlessness, concentration difficulties, inattention and hectic/fidgety behaviour. Reactive aggression was measured using four items referring to aggression in response to being insulted, teased, not getting something and having something taken. Proactive aggression was measured using four items measuring scaring others to force them to do something, bossing others around, humiliating others and using threats to get something. The administration and response format of the youth SBQ was the same as that for the teachers.

2.3. Statistical procedure

The exacerbation and attenuation hypotheses can be operationalised as the moderation of a cross-lagged effect of ADHD symptoms on (reactive or proactive) aggression by anxiety. We tested this by fitting 8-wave (for the teacher data) and 3-wave (for the self-report data) cross-lagged panel models (CLPMs), including ADHD symptoms by anxiety interaction terms. We did this separately for reactive and proactive aggression. An example Mplus file is provided in Supplementary Materials.

Given that we had multiple indicators of each construct we could, in principle have specified each as a latent variable in these tests; however, in practice latent interactions are computationally demanding and our hypotheses required testing 7 (for the teacher data) or 2 (for the self-report data) interactions per model. As a pragmatic solution to this problem, we used a two-step approach. Here, factor scores for each construct were estimated in step 1 and a cross-lagged panel model treating these as observed was estimated in step 2. Factor scores were estimated from the joint measurement model of the three constructs (ADHD symptoms, anxiety and reactive or proactive aggression) over the available waves of data. All latent factors were allowed to correlate with one another within and across time points, residual covariances between items measured at different time points were freely estimated, and latent factor variances were fixed to 1 for scaling and identification. Factor score determinacies (an estimate of the correlation between factor scores and the underlying latent variable) in step 1 were examined to ensure the quality of factor scores as proxies for the relevant latent variables. Ideally factor score determinacies should be >0.90 (Gorsuch, 1983). In step 2, the CLPM model included all autoregressive and cross-lagged paths and within-wave (residual) covariances. Both step 1 (measurement) and step 2 (CLPM) models were estimated i*n Mplus 7.4* using maximum likelihood estimation (Muthén and Muthén, 2014).

Finally, given that sex differences in ADHD symptoms, anxiety and aggression have all previously been observed (e.g. Archer, 2004; Gershon and Gershon, 2002; McLean and Anderson, 2009), we estimated the models first in the whole sample and then stratified by gender. Gender-stratified analyses were conducted using a multi-group model with no cross-group equality constraints on any parameters.

3. Results

3.1. Reactive aggression

3.1.1. Observer (teacher) reports

In step 1, a single-factor measurement model for both anxiety and reactive aggression was supported at each time point. For ADHD symptoms, there was differentiation of ADHD into an attention-deficit and a hyperactivity/impulsivity factor for the first three waves and here we specified ADHD as a higher-order construct with attention-deficit and hyperactivity/impulsivity as lower order factors. At the subsequent waves we specified ADHD symptoms as unidimensional. We estimated factor scores from the joint measurement model including all of these latent variables correlated across and within time points. All factor score determinacies were all >0.90; however, many of the factor scores had positively skewed distributions, sometimes >1. As positively skewed outcome variables can promote the spurious detection of interactions, we transformed all variables to normality prior to fitting the CLPM. While this does not guarantee unbiased estimates of interaction parameters, it reduces the likelihood that a detected interaction is a mere artefact of scaling (Murray, Molenaar, et al., 2016). We used a Box-cox transformation to obtain as close as an approximation to normality as possible for each variable. Following this transformation all skewness values were <|0.15|. The correlation matrix for these factor scores is provided in the appendix.

In step 2, we used the Box-Cox transformed factor scores to fit a cross-lagged panel model as shown in Figure 1 which provides the resulting standardised parameter estimates. For clarity, only paths pertaining directly to the exacerbation and attenuation hypotheses are shown. Waves are labelled according to the approximate age of the youths at time of assessment and statistically significant parameters (p<0.05) italicised. Full model results are provided in Table 1 of Supplementary Materials. As the gender-stratified results were highly similar to the combined sample analyses, we focus only on the latter here.

According to these, ADHD symptoms were highly stable across time while anxiety and reactive aggression were overall moderately stable. The main effects of anxiety on reactive aggression were generally small, variable in direction, and statistically significant only about half the time. The main effects of ADHD symptoms on reactive aggression were, in contrast, consistently positive, statistically significant and greater than $\beta = 0.10$. There was only one significant interaction between anxiety and ADHD symptoms. This was between ages 10 and 11. The nature of the interaction was that as anxiety increased, the effect of ADHD symptoms on reactive aggression decreased. For example, based on computing simple slopes, at 1SD below the mean on anxiety, the effect of ADHD symptoms would be expected to be $\beta = 0.16$ while at 1SD above the mean on anxiety, the effect would be expected to be $\beta = 0.08$. This is in line with the attenuation hypothesis and, therefore, runs counter to our hypothesis that exacerbation - not attenuation – should describe the role of anxiety in ADHD symptoms-reactive aggression relations.

3.1.2. Self-report (youth) measures

Single-factor measurement models for each construct were supported across the three waves of data for which self-report data was available. The determinacies of the factor scores estimated from these models were >0.90 for ADHD symptoms but as low as 0.86 for the other constructs. Though not ideal, we judged this to be close enough to .90 to proceed with a two-step approach. Box-cox transformation brought the skewness of all factor scores <|0.05|. The correlation matrix for these factor scores is provided in the Supplementary Materials.

The standardised parameter estimates pertaining to the exacerbation and attenuation hypotheses from the CLPM fit using these factor scores are provided in Figure 2. Combined sample and gender-stratified results were highly similar thus we discuss only the combined sample analyses, with gender-stratified analyses provided in Supplementary Materials (Table

2). According to these, ADHD symptoms, anxiety and reactive aggression were highly stable, especially ADHD symptoms. The main effects of both anxiety and ADHD symptoms on reactive aggression were negative and small but statistically significant. There were no significant interactions between anxiety and ADHD symptoms. Thus, our hypothesis that anxiety should exacerbate the effect of ADHD symptoms on reactive aggression was not supported by youth self-reports.

3.2. Proactive aggression

3.2.1. Observer (teacher) reports

In step 1, the measurement models for anxiety and ADHD symptoms were specified as reported for the reactive aggression. A single factor model was also supported for proactive aggression. The factor scores from the joint measurement model of these constructs estimated all had determinacies >0.90 and positive skewness. The same Box-cox transformation procedure as described above was implemented and reduced all skewness values to <|0.20|. The correlation matrix for these factor scores is provided in the appendix.

Standardised parameter estimates from the CLPM estimated in step 2 using these factor scores are provided in Figure 3. Again, for clarity, only those pertaining to the exacerbation and attenuation hypotheses are shown with full model results provided in Table 3 of Supplementary Materials. The gender-stratified results (see Table 3) were again highly similar to the combined sample analyses and we thus focus on the combined sample results here. According to these, proactive aggression was moderately stable over time with some variation in exactly how stable. Four of the seven cross-lagged effects of anxiety on proactive aggression were statistically significant and around β=-0.10. All of the cross-lagged effects of ADHD symptoms on proactive aggression were statistically significant and positive. There were, however, no significant interactions between anxiety and ADHD symptoms.

3.2.2. Youth self-reports

In step 1, the measurement models for anxiety and ADHD symptoms were specified as reported for the reactive aggression. A unidimensional measurement model was also supported for proactive aggression. All factor score determinacies were again close to or greater than 0.90 (minimum=0.86 for Anxiety at age 11). Box-cox transformation brought the skewness of factor scores all under |0.12|. The correlation matrix for these factor scores is provided in the appendix.

Parameter estimates pertaining to the exacerbation and attenuation hypotheses from the CLPM using these factor scores are provided in Figure 4. Full model results are provided in Supplementary Materials. Combined sample and gender-stratified results (Table 4 of Supplementary Materials) were very similar and we, therefore, focus on the former results here. According to these, proactive aggression was highly stable, although slightly less so than reactive aggression. The main effects of anxiety on proactive aggression were negative and small but statistically significant. There were, however, no significant cross-lagged effects of ADHD symptoms on proactive aggression nor any significant ADHD symptoms by anxiety interactions.

4. Discussion

In this study, we aimed to contribute to helping to explain the previous uncertainties surrounding the question of whether anxiety exacerbates or attenuates aggression associated with ADHD symptoms. Specifically, we proposed and tested the hypothesis that anxiety exacerbates reactive aggression but attenuates proactive aggression associated with ADHD symptoms. There was no evidence for either hypothesis. In fact, the only moderating effect of anxiety was attenuating the effect of ADHD symptoms on *reactive aggression*: exactly counter to our predictions and limited to just one point in time. Our results suggest that rather

than indirectly affecting aggression in ADHD symptom by moderating ADHD symptom-aggression links, anxiety has a direct protective effect against both reactive and proactive aggression.

A number of previous studies have reported an association between ADHD and reactive aggression (e.g. Card and Little, 2006). While this has generally been interpreted as ADHD symptoms being causal in reactive aggression, a lack of studies with repeated measures of both constructs (or other appropriate design) have made direction of causation difficult to confirm. Our teacher-reported results showing a significant and positive cross-lagged effects of ADHD on reactive aggression at all waves, therefore, provide important evidence bolstering this interpretation. However, this was not replicated in the youth self-reports which showed no significant cross-lagged effect of ADHD symptoms on reactive aggression. This latter result may reflect the unreliability of self-reported ADHD symptoms. It has been noted that ADHD symptom self-reports are liable to underestimate symptoms (e.g. Sibley et al., 2012)

Past research has provided a much more inconsistent picture regarding the relation between ADHD symptoms and proactive aggression. Bennet et al. (2004) proposed that proactive aggression could be correlated with ADHD because those with ADHD and reactive aggression may associate with delinquent peers from whom they 'pick up' proactive aggression behaviours. They reported a positive ADHD-proactive aggression association in 12 to 15 year olds, consistent with their hypothesis. However, others have found contradictory evidence, consistent with the idea that proactive aggression is less common among individuals with high levels of ADHD symptoms. Vitaro et al. (2002), for example, found that children exhibiting both proactive and reactive aggression showed fewer inattention symptoms than those exhibiting reactive aggression alone. Similarly, Retz and Rözler (2010) found a negative association between ADHD symptoms and proactive

aggression in a forensic sample. Our teacher reported results indicated significant *positive* cross-lagged effects of ADHD symptoms on proactive aggression across all waves. However, this does not necessarily contradict the previous findings of a negative association: there may be negative association between ADHD symptoms and proactive aggression conditional on the presence of aggressive behaviour (or high risk thereof) but a positive association in the population as a whole. Thus, the negative association should be observed only in high (aggression) risk samples such as the forensic sample of Retz and Rözler (2010) or when considering the association specifically within the aggressive children of a normative sample (as in Vitaro et al., 2002). Such a pattern could indicate that those with lower levels of ADHD symptoms are more adept at channelling aggressive tendencies in a goal-directed manner, thus manifesting their aggression instrumentally rather than impulsively. However, the positive effect of ADHD on proactive aggression in the current study was again limited to teacher reports: youth self-reports suggested no significant cross-lagged effects of ADHD on proactive aggression. As with the lack of association between reactive aggression and ADHD symptoms, this may reflect under-reporting of ADHD symptoms in self-reports.

With regards to the effect of anxiety, we found that within waves, it was generally positively correlated with both proactive and reactive aggression across both teacher and youth self-reports. Previous research has suggested that anxiety and reactive aggression are positively correlated due to the presence of shared neurobiological features such as autonomic hyperarousal and hypervigilance to threatening stimuli (Bubier and Drabick, 2009). Similarly, at the cognitive level, anxiety is thought to be associated with hostile attributional biases that can drive aggression, especially of the reactive type (e.g. Marsee et al., 2008). However, we found significant and negative cross-lagged effects on both reactive aggression (according to youth self-reports but with only limited support in teacher reports) and proactive aggression (according to youth self-reports and largely supported in teacher

reports). Thus, in terms of the causal effect of anxiety on aggression, our results support the view that anxiety acts primarily as an inhibiting force against maladaptive, even implusive aggression (e.g. Bloemsma et al., 2013; Tremblay et al., 1994; Pliska, 1989).

In contrast to both the exacerbation and attenuation hypotheses, the effect of anxiety on both reactive and proactive aggression was effectively limited to a main effect. The only evidence of a moderating effect of anxiety was limited to one of 18 paths tested and was in the opposite direction to that predicted. Thus, our results fairly unambiguously failed to support the hypothesis that anxiety exacerbates reactive aggression and attenuates proactive aggression.

A notable feature of the current results were the discrepancies between results according to informant (teacher) reports and youth self-reports. This is a common occurrence in psychopathology research (e.g. Achenbach, 2006). Neither source can be expected to be uniformly more accurate than the other. One the one hand, (non-self) informants may have difficulty identifying psychopathologies that manifest primarily as internal states including the anxiety symptoms that were a major focus of the current study. They may also find it difficult to distinguish reactive and proactive aggression because they do not have direct access to the motivations and emotions underlying aggressive acts (e.g. Vitaro et al., 2002). In addition, teachers typically see the target in one context (school) only, whereas behaviour may vary across contexts (e.g. De Los Reyes, 2011). Further, teacher ratings have been shown to be vulnerable to halo effects, i.e. when individuals who display one behaviour are incorrectly rated as displaying a second, conceptually related behaviour (e.g. Hartung et al., 2010). These effects have previously been identified in teacher ratings of ADHD (e.g. Jackson and King, 2004; Schachar et al., 1986). However, self-reports are vulnerable to their own sets of biases; some unique, some overlapping with those of informant reports. These can include socially desirable responding (Lanya and Wershba, 2013) and responding

according to implicit theories about psychopathology (Lahey et al., 2012) and in younger respondents, issues of comprehension of the items and the concepts to which they refer. These concerns and the fact that the current results differed in many cases across raters underlines the importance of obtaining data from multiple sources when attempting to make inferences regarding the inter-relations and interactions among different symptoms.

4.1. Limitations

It is also important to consider the limitations of the current study. The primary limitation is that only brief measures of ADHD, anxiety and reactive and proactive aggression were available. Although factor score determinacies were generally good, more comprehensive measures would nonetheless provide more robust tests of our hypotheses. Second, attempts to compare effects on proactive versus reactive aggression are complicated by the fact that they are strongly correlated. While, in principle, this can be addressed via methods such as utilising a bi-factor measurement model to separate variation common versus unique to these forms of aggression, in practice in the case of the measure used in the current sample, this leaves insufficient reliable variance in the unique factors (e.g. Reise, 2012). Third, an additional informant with clinical expertise or peer informants could have been useful in potentially resolving the discrepancies between youth and teacher reports. Finally, we did not have comprehensive data on the use of medications for ADHD and anxiety and were thus unable to control for their effects. Based on a self-report medication use item, we estimate that around 5% of the sample may have taken medications typically prescribed for ADHD symptoms.

5. Conclusions

Our results suggest that anxiety protects against proactive aggression and likely also reactive aggression. However, it appears to neither exacerbate nor attenuate the effect of ADHD symptoms on these outcomes.

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Figures

Figure 1

Cross-lagged panel model of ADHD, anxiety and reactive aggression according to teacher reports

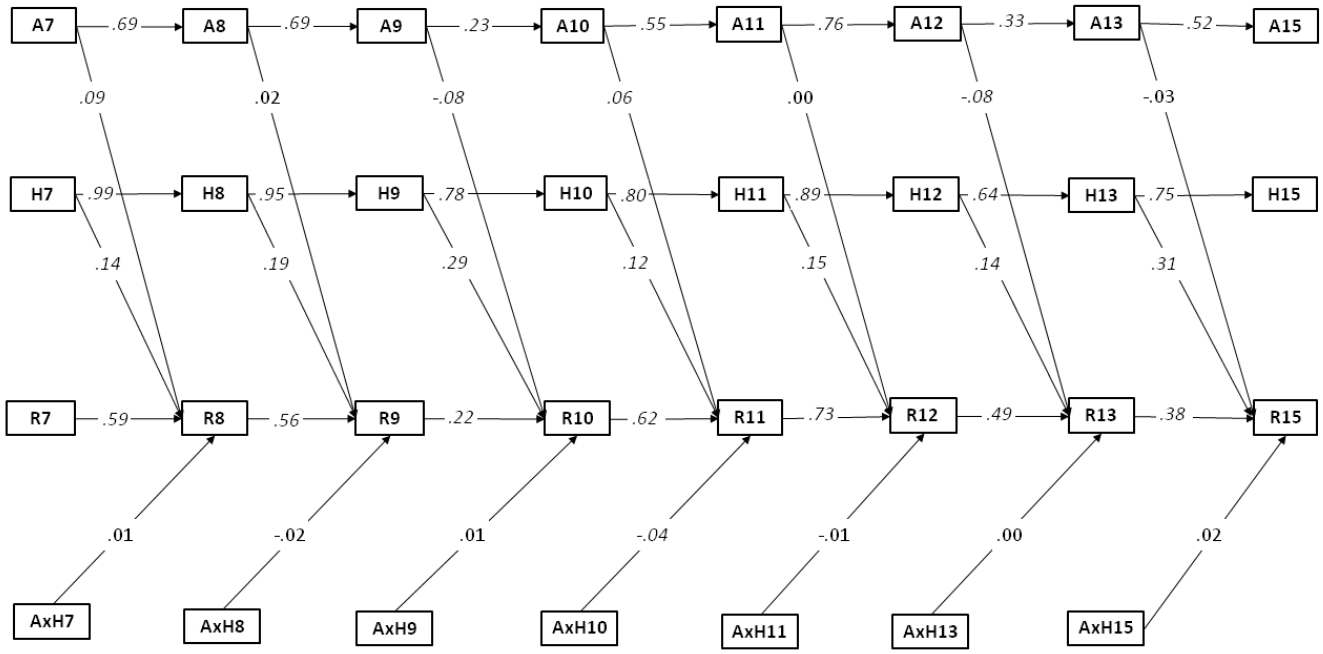

Figure 2

Cross-lagged panel model of ADHD, anxiety and reactive aggression according to self-reports

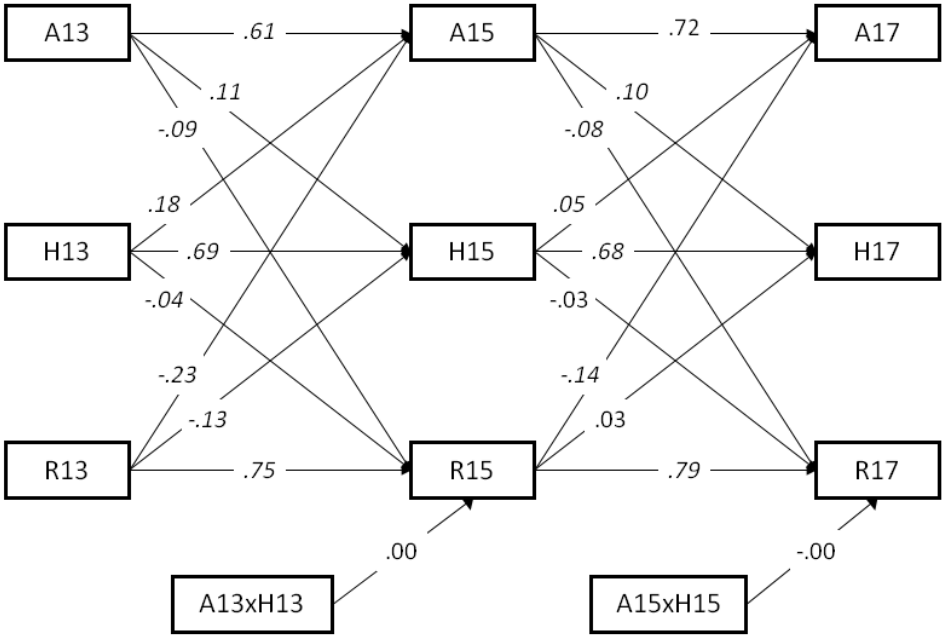

Figure 3

Cross-lagged panel model of ADHD, anxiety and proactive aggression according to teacher reports

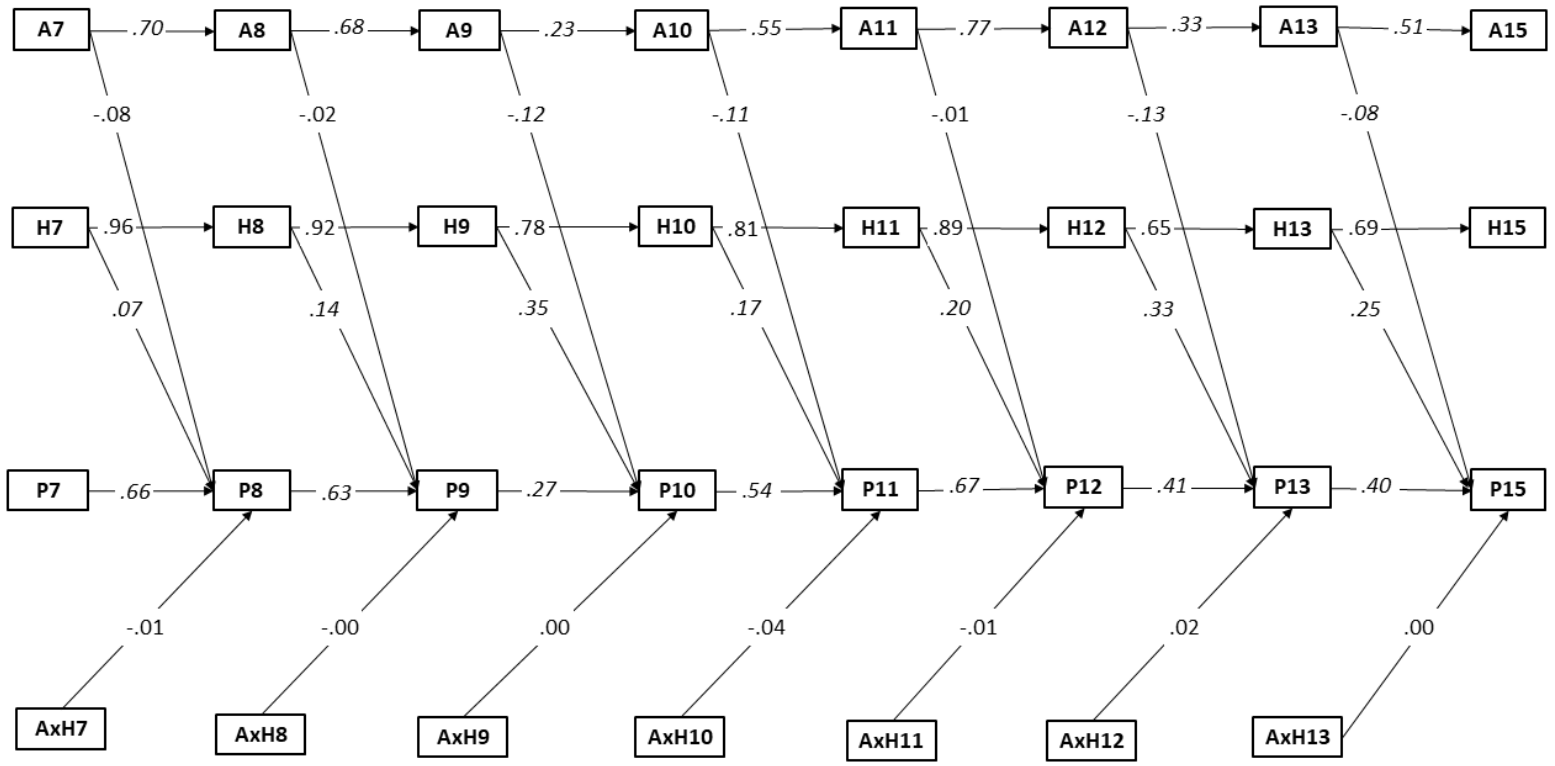

Figure 4

Cross-lagged panel model of ADHD, anxiety and proactive aggression according to self-reports

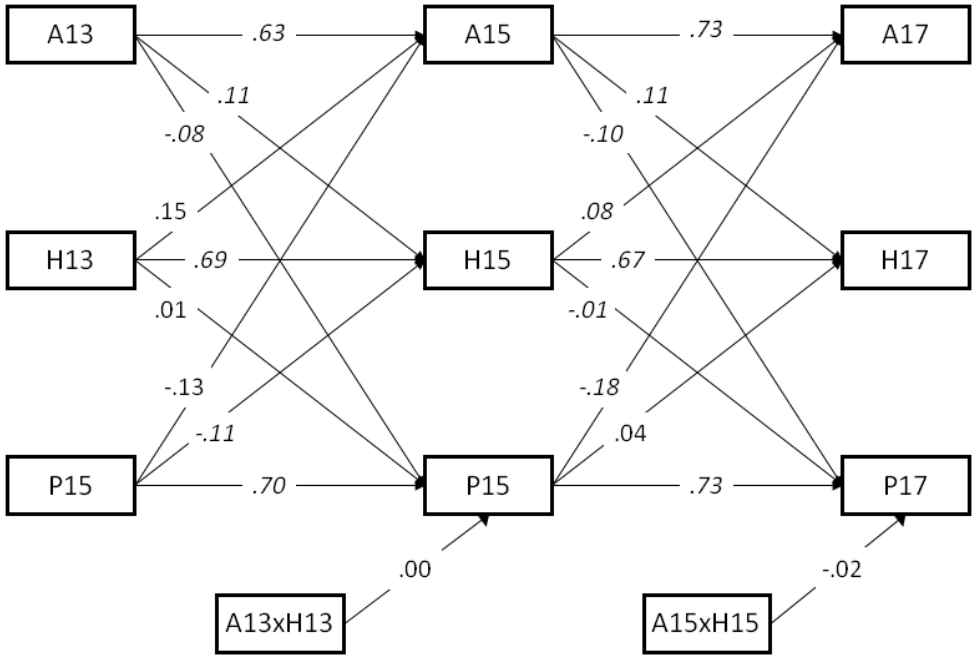

Figure Notes

Figure 1: *Note.* Figure shows the cross-lagged panel model for teacher-reported anxiety (A), ADHD (H) and reactive aggression (R) based on factor scores transformed to normality. For clarity, only the parameters of primary relevance to the exacerbation/attenuation hypotheses are shown, with full model parameters provided in Table 1 of Supplementary Materials. The numbers 13, 15 and 17 represent the median ages of participants at the relevant measurement wave. Product terms are indicated as the multiplication of anxiety at a given measurement wave with ADHD at the same measurement wave. For example, A13xH13 indicates an interaction between age 13 anxiety and ADHD. Statistically significant parameters (p<.05) are indicated in italics.

Figure 2. Figure shows the cross-lagged panel model for self-reported anxiety (A), ADHD (H) and reactive aggression (R) based on factor scores transformed to normality. For clarity, only the parameters of primary relevance to the exacerbation/attenuation hypotheses are shown, with full model parameters provided in Table 2 of Supplementary Materials.

Figure 3. Figure shows the cross-lagged panel model for teacher-reported anxiety (A), ADHD (H) and proactive aggression (P) based on factor scores transformed to normality. For clarity, only the parameters of primary relevance to the exacerbation/attenuation hypotheses are shown, with full model parameters provided in Table 3 of Supplementary Materials.

Figure 4. Figure shows the cross-lagged panel model for self-reported anxiety (A), ADHD (H) and proactive aggression (P) based on factor scores transformed to normality. For clarity, only the parameters of primary relevance to the exacerbation/attenuation hypotheses are shown, with full model parameters provided in Table 4 of Supplementary Materials.